

Dynamic Behavior of a Brucellosis Infection Model with the Effect of Infected Dogs

Yaoyao Kang, Xamxinur Abdurahman

Abstract—Brucellosis is one of the major public health problems in China. According to relevant literature, dogs with brucellosis can not only infect other dogs, but also easily infect human and also release pathogenic bacteria into the environment. In this paper, we proposed a dynamical model of sheep-dog-human brucellosis transmission. At the same time, we considered the environmental bacterial infections of sheep, dog and human in our model. The global asymptotic behavior of model is determined by the size of the basic reproduction number R_0 . If $R_0 < 1$, the disease-free equilibrium is global asymptotically stable. If $R_0 > 1$, the model is uniformly persistent and the endemic equilibrium is global asymptotically stable.

Index Terms—Brucellosis, basic reproduction number, infected environment, global stability.

I. INTRODUCTION

BRUCELLOSIS, also known as Rock fever, is one of the zoonotic diseases caused by different species of Brucella[1]. Brucellosis is generally result in by *B.abortus* in cattle, *B.melitensis* in sheep and goats, *B.ovis* in small ruminant, *B.suis* in pig and *B.canis* in dog[2]. So it is prevalent among sheep, cattle, dog and pig. In animals, there are two ways of brucellosis infection: one is through direct contact with infected animals or the feces, the second is through the indirect transmission caused by the contact with the infected environment such as soil, contaminated water etc. The survival time of brucella differs from one to four months in the contaminated soil and water, and about two months in milk and meat. However, it is easily killed by direct sunlight, high temperature and effective disinfectant[3]. In the initial stage of infection, there are no obvious clinical symptoms and it is difficult to diagnose[4]. Human brucellosis infection is mainly caused by occupational exposure to infected livestock, ingestion of unpasteurised dairy products and contaminated meats[5]. The infection through laboratory exposure among veterinary students and laboratory workers also attracts much attention in recent years. There is also some reports on the human to human transmission cases [6]. Therefore, as long as the disease is eradicated from susceptible animals, the infection of human brucellosis can be greatly reduced[7].

In China, the number of human brucellosis cases has increased dramatically since 2000, and materials show that brucellosis is widely distributed in the northern and western parts of China. With the legal exhibition of animal husbandry production, Xinjiang Uyghur Autonomous Region is known

as the epidemic area of human brucellosis in China[8]. At present, sheep brucellosis is very common. The documents on the dog brucellosis infection also show that the overall prevalence rate of canine Brucellosis in Urumqi is about 25 percent, besides, the brucella of canine and ovine species coexists poses a severe threat to public health. Among the different groups, the dog brucellosis prevalence in pastoral areas was the highest[9]. According to an US report, the brucellosis prevalence of dogs is higher among stray dogs and free-roaming dogs[10]. In the early 20th century, reports confirm human brucellosis patients in China. Two human brucellosis cases were first reported in Chongqing province in 1905 and a human brucellosis case was reported in Fujian province in 1916 [11]. Human brucellosis is not fatal, almost negligible, but the disease can last for several years[12]. It has been suggested that the incidence of human brucellosis will be correspondingly and significantly reduced when using vaccines and slaughter to control livestock brucellosis. Nowadays, strict quarantine in the animal industry, combined with slaughter policies and alternative vaccine strategies, has been shown to be necessary to effectively prevent and control the spread of brucellosis in China[13].

Mathematical modelling has turned into an important tool for the comprehension of transmission dynamics of epidemic diseases and to propose control strategies for the infectious diseases, see for example[1-5,8,11,14-18]. Modeling real world epidemics can be a challenging and complex task and its usefulness is undisputable, because the more realistic the modelling is, the more it can contribute to a better understanding of the physical phenomenon itself[14]. Recently, many researchers have made great achievements in the study of brucellosis. For instance, Hou et al. [1] established a dynamic model with the sheep-human transmission of brucellosis. Nie et al. [15] proposed a dynamical model of cattle brucellosis with Susceptible-Exposed-Infected-Virus. Zinsstag et al. [16] studied a dynamic model of cattle-sheep-human with seropositive and immunized groups. Li M.T. et al. [11] set up a four-dimensional staged brucellosis model. Considering the current situation of dog brucellosis infection, we propose an eight-dimensional model of sheep-dog-human brucellosis transmission.

This paper is organized as follows. In Section 2, we construct the model and get the basic reproduction number R_0 . In Section 3, we analyze the global asymptotic stability of the disease-free equilibrium. In Section 4, we demonstrate the existence and global asymptotic stability of the endemic equilibrium. In Section 5, some numerical simulation will be used to verify our analytical results. Finally, we give some discussions.

This work was supported by National Natural Science Foundation of China(Grant Numbers 11861063,11771373).

Yaoyao Kang and Xamxinur Abdurahman are with College of Mathematics and System Sciences, Xinjiang University, Urumqi, Xinjiang, P.C. 830046 China.

Corresponding author: Xamxinur Abdurahman, e-mail: xamxinur@sina.com.

II. DYNAMIC MODEL

Based on the facts of brucellosis infection in the sheep farms, we divided the sheep population into three compartments: the susceptible, the infectious and the vaccinated compartment which is denoted by $S(t), I(t), V(t)$. At the same time, based on the characteristics of brucellosis transmission in dogs, we can classify the dog population into two compartments: the susceptible S_d and the infectious I_d . Human population is also divided into two groups: susceptible S_h and infected I_h . As is known to all, infected sheep and dog generates infection in two way: the direct and indirect modes of transmission. We defined the average number of brucella to be sufficient to infect the host as an infected unit. So we use $W(t)$ to denote the brucella in the environment at time t . So a brucellosis model can be given as a system of ordinary differential equations as follows:

$$\begin{cases} \frac{dS}{dt} = A_1 - \beta SI - \nu S - \phi SW - d_1 S \\ \frac{dI}{dt} = \beta SI + \phi SW - (d_1 + \alpha) I \\ \frac{dV}{dt} = \nu S - d_1 V \\ \frac{dW}{dt} = kI - (\mu + n\tau)W + \lambda I_d \\ \frac{dS_d}{dt} = A_2 - \phi_d S_d W - d_2 S_d \\ \frac{dI_d}{dt} = \phi_d S_d W - (d_2 + c) I_d \\ \frac{dS_h}{dt} = A_3 - \beta_h S_h I - \phi_h S_h W - \beta_d S_h I_d + \gamma I_h - d_3 S_h \\ \frac{dI_h}{dt} = \beta_h S_h I + \phi_h S_h W + \beta_d S_h I_d - \gamma I_h - d_3 I_h \end{cases} \quad (1)$$

All the parameters are considered is nonnegative in system (1), which are described in Table 1.

Because the first six equations are independent of the last two equations, we just need to analyze the subsystem composed of the first six equations, as shown in the following model:

$$\begin{cases} \frac{dS}{dt} = A_1 - \beta SI - \nu S - \phi SW - d_1 S \\ \frac{dI}{dt} = \beta SI + \phi SW - (d_1 + \alpha) I \\ \frac{dV}{dt} = \nu S - d_1 V \\ \frac{dW}{dt} = kI - (\mu + n\tau)W + \lambda I_d \\ \frac{dS_d}{dt} = A_2 - \phi_d S_d W - d_2 S_d \\ \frac{dI_d}{dt} = \phi_d S_d W - (d_2 + c) I_d \end{cases} \quad (2)$$

The initial condition for system (2) is $S(0) > 0, I(0) > 0, V(0) > 0, W(0) > 0, S_d(0) > 0, I_d(0) > 0$. It is obvious that system (2) exists unique positive solution satisfies given initial condition. Adding up the first three equations, we can find that

$$\frac{d(S + I + V)}{dt} = A_1 - d_1(S + I + V) - \alpha I$$

it follows that

$$\limsup_{t \rightarrow \infty} (S + I + V) \leq \frac{A_1}{d_1} \quad (3)$$

TABLE I
DESCRIPTIONS OF PARAMETERS IN SYSTEM (1)

parameters	comments
A_1	the input number of sheep
A_2	the input number of dog
A_3	the input number of human
β	sheep-to-sheep transmission rate
β_h	transmission rate from sheep to human
β_d	transmission rate from dog to human
ϕ	transmission rate of contaminated environment to susceptible sheep
ϕ_d	transmission rate of contaminated environment to susceptible dog
ϕ_h	transmission rate of contaminated environment to susceptible human
ν	sheep vaccination rate
d_1	the natural death rate of sheep
d_2	the natural death rate of dog
d_3	the natural death rate of human
μ	the natural decaying rate of brucella in the environment
n	disinfection times
τ	the efficient disinfection rate
λ	Brucella shedding rate of infectious dog to the environment
k	Brucella shedding rate of infectious sheep to the environment
c	the disease-related elimination rate of dog
α	the disease-related elimination rate of sheep
γ	human recovery rate

Combining the fifth and sixth equations of system (2) yields

$$\begin{aligned} \frac{d(S_d + I_d)}{dt} &= A_2 - d_2(S_d + I_d) - cI_d \\ &\leq A_2 - d_2(S_d + I_d) \end{aligned}$$

then it follows that

$$\limsup_{t \rightarrow \infty} (S_d + I_d) \leq \frac{A_2}{d_2} \quad (4)$$

From the fourth equation of system (2) we have

$$\frac{dW}{dt} \leq \frac{kA_1 d_2 + \lambda A_2 d_1}{d_1 d_2} - (\mu + n\tau)W$$

Let $p = \max\{k, \lambda\}$, then we have

$$\limsup_{t \rightarrow \infty} W \leq p \frac{A_1 d_2 + A_2 d_1}{d_1 d_2 (\mu + n\tau)} \quad (5)$$

By equations (3)-(5), we can conclude that

$$\begin{aligned} X = \{ &(S, I, V, W, S_d, I_d) \mid S, I, V, W, S_d, I_d \geq 0, \\ &0 \leq S + I + V \leq \frac{A_1}{d_1}, 0 \leq S_d + I_d \leq \frac{A_2}{d_2}, \\ &0 \leq W \leq p \frac{A_1 d_2 + A_2 d_1}{d_1 d_2 (\mu + n\tau)} \} \end{aligned}$$

is the positively invariant set respect to system (2). It is evident that system (2) has a disease-free equilibrium $P_0 = (S^0, 0, V^0, 0, S_d^0, 0)$, where

$$S^0 = \frac{A_1}{d_1 + \nu}, V^0 = \frac{A_1 \nu}{(d_1 + \nu)(d_1 + \alpha)}, S_d^0 = \frac{A_2}{d_2}$$

Following the method of Van den Driessche and Watmough [19], we have

$$F = \begin{pmatrix} \beta S^0 & \phi S^0 & 0 \\ 0 & 0 & 0 \\ 0 & \phi_d S_d^0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} d_1 + \alpha & 0 & 0 \\ -k & \mu + n\tau & -\lambda \\ 0 & 0 & d_2 + c \end{pmatrix}$$

Therefore, the next generation matrix is

$$FV^{-1} = \begin{pmatrix} A_{11} & A_{12} & A_{13} \\ 0 & 0 & 0 \\ A_{31} & A_{32} & A_{33} \end{pmatrix}$$

The characteristic polynomial of FV^{-1} is

$$P(\Lambda) = \Lambda[\Lambda^2 - (A_{11} + A_{33})\Lambda + (A_{11}A_{33} - A_{13}A_{31})]$$

So the basic reproduction number of system (2) is defined as following

$$R_0 = \frac{A_{11} + A_{33} + \sqrt{(A_{11} - A_{33})^2 + 4A_{13}A_{31}}}{2}$$

where

$$A_{11} = \frac{\beta S^0}{d_1 + \alpha} + \frac{\phi S^0 k}{(\mu + n\tau)(d_1 + \alpha)}$$

$$A_{13} = \frac{\lambda \phi S^0}{(d_2 + c)(\mu + n\tau)}$$

$$A_{31} = \frac{\phi_d S_d^0 k}{(d_1 + \alpha)(\mu + n\tau)}$$

$$A_{33} = \frac{\phi_d S_d^0 \lambda}{(d_2 + c)(\mu + n\tau)}$$

III. GLOBAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM

In this section, we demonstrate the global stability of the disease-free equilibrium.

Let $M = F - V$, we have

$$M = \begin{pmatrix} \beta S^0 - (d_1 + \alpha) & \phi S^0 & 0 \\ k & -(\mu + n\tau) & \lambda \\ 0 & \phi_d S_d^0 & -(d_2 + c) \end{pmatrix}$$

Define $s(M) = \max\{Re\Lambda : \Lambda \text{ is an eigenvalue of } M\}$, with $s(M)$ is a simple eigenvalue of M with a positive eigenvector, by the Theorem 2[19], we have

$$R_0 > 1 \Leftrightarrow s(M) > 0, R_0 < 1 \Leftrightarrow s(M) < 0.$$

For the disease-free equilibrium of system (2), the following results can be established:

Theorem 3.1 The disease-free equilibrium P_0 is locally asymptotically stable when $R_0 < 1$, and unstable when $R_0 > 1$.

Proof: It's obvious that the hypothesis (A1-A4) of Lemma 2 in [19] is satisfied. Next we verify hypothesis (A5), thus we only need to prove

$$J|_{P_0} = \begin{pmatrix} M & 0 \\ J_3 & J_4 \end{pmatrix}$$

have negative real parts, where $J_3 = -F$.

$$J_4 = \begin{pmatrix} -d_1 - \nu & 0 & 0 \\ \nu & -d_1 & 0 \\ 0 & 0 & -d_2 \end{pmatrix}$$

By calculating the eigenvalues of J_4 , we have $s(J_4) = \max\{-d_1, -(d_1 + \nu), -d_2\} < 0$. Therefore, if $R_0 < 1$, then $s(M) < 0$ and $s(J|_{P_0}) < 0$, and the disease-free equilibrium P_0 of system (2) is locally stable. If $R_0 > 1$, then $s(M) > 0$, which means that P_0 is unstable.

Theorem 3.2 The disease-free equilibrium P_0 of system (2) is globally asymptotically stable when $R_0 < 1$.

Proof: Firstly, notice that the condition $R_0 < 1$ implies $A_{11} \leq 1, A_{33} \leq 1$, and $\frac{-P(1)}{A_{13}} < 0$.

Consider a Lyapunun function as follows:

$$L_1 = a_1(S - S^0 - S^0 \ln \frac{S}{S^0}) + a_2(V - V^0 - V^0 \ln \frac{V}{V^0})$$

$$+ a_3(S_d - S_d^0 - S_d^0 \ln \frac{S_d}{S_d^0}) + a_4I + a_5W + a_6I_d$$

where

$$a_1 = a_2 = a_4 = \frac{1 - A_{33}}{(d_1 + \alpha)A_{13}}$$

$$a_3 = a_6 = \frac{\phi_d S_d^0}{(\mu + n\tau)(d_1 + \alpha)}$$

$$+ \frac{\lambda}{d_2 + c} \left(\frac{(1 - A_{33})\phi S^0}{A_{13}(d_1 + \alpha)(\mu + n\tau)} \right)$$

$$a_5 = \frac{(1 - A_{33})\phi S^0}{A_{13}(d_1 + \alpha)(\mu + n\tau)} + \frac{\phi_d S_d^0}{(\mu + n\tau)(d_1 + \alpha)}$$

Then the derivative of L_1 along with the solution of system (2) is

$$\frac{dL_1}{dt} = a_1(1 - \frac{S^0}{S})(A_1 - \beta SI - \nu S - \phi SW - d_1 S)$$

$$+ a_3(1 - \frac{S_d^0}{S_d})(A_2 - \phi_d S_d W - d_2 S_d)$$

$$+ a_2(1 - \frac{V^0}{V})(\nu S - d_1 V) + a_6(\phi_d S_d W$$

$$- (d_2 + c)I_d) + a_4(\beta SI + \phi SW - (d_1 + \alpha)I)$$

$$+ a_5(kI - (\mu + n\tau)W + \lambda I_d)$$

$$= a_1[A_1(S - S^0)(\frac{1}{S} - \frac{1}{S^0}) + \nu(V - V^0)(\frac{S}{V} - \frac{S^0}{V^0})]$$

$$+ a_4(\beta SI + \phi SW - (d_1 + \alpha)I)$$

$$+ a_3A_2(S_d - S_d^0)(\frac{1}{S_d} - \frac{1}{S_d^0})$$

$$+ a_5(kI - (\mu + n\tau)W + \lambda I_d)$$

$$+ a_6(\phi_d S_d W - (d_2 + c)I_d)$$

$$- a_1(S - S^0)(\beta I + \phi W) - a_3(S_d - S_d^0)\phi_d W$$

$$= a_1 d_1 V^0 (3 - \frac{V}{V^0} - \frac{SV^0}{VS^0} - \frac{S^0}{S})$$

$$+ a_1 d_1 S^0 (2 - \frac{S^0}{S} - \frac{S}{S^0}) - a_1(S - S^0)\beta I$$

$$+ a_1(\beta SI + \phi SW - (d_1 + \alpha)I)$$

$$+ a_3(\phi_d S_d W - (d_2 + c)I_d)$$

$$+ a_3A_2(S_d - S_d^0)(\frac{1}{S_d} - \frac{1}{S_d^0})$$

$$+ \frac{d_2 + c}{\lambda} a_3(kI - (\mu + n\tau)W + \lambda I_d) - a_1(S - S^0)\phi W - a_3(S_d - S_d^0)\phi_d W$$

After a careful calculation, it yields that

$$\begin{aligned} \frac{dL_1}{dt} = & a_1 d_1 S^0 \left(2 - \frac{S^0}{S} - \frac{S}{S^0}\right) \\ & + a_1 d_1 V^0 \left(3 - \frac{V}{V^0} - \frac{SV^0}{VS^0} - \frac{S^0}{S}\right) \\ & + a_3 A_2 (S_d - S_d^0) \left(\frac{1}{S_d} - \frac{1}{S_d^0}\right) - \frac{P(1)}{A_{13}} I. \end{aligned}$$

So, when $R_0 < 1$, $\frac{dL_1}{dt} < 0$, and $\frac{dL_1(t)}{dt} = 0$ holds if and only if $S = S^0, I = 0, V = V^0, W = 0, S_d = S_d^0, I_d = 0$. Thus the disease-free equilibrium P_0 is global asymptotically stable by LaSalle's Invariance Principle [20]. This completes the proof.

IV. THE GLOBAL STABILITY OF ENDEMIC EQUILIBRIUM

In this section, we firstly study the uniformly persistence of the system (2), then prove the existence and the global stability of endemic equilibrium.

Define $X_0 = \{(S, I, V, W, S_d, I_d) \in X \mid I, W, I_d > 0\}$ and $\partial X_0 = X \setminus X_0$.

Theorem 4.1 When $R_0 > 1$, there exists a positive constant ε_1 such that when $|I(0)| < \varepsilon_1, |W(0)| < \varepsilon_1, |I_d(0)| < \varepsilon_1$ for $(S(0), I(0), V(0), W(0), S_d(0), I_d(0)) \in X_0$,

$$\limsup_{t \rightarrow \infty} \max\{I(t), W(t), I_d(t)\} > \varepsilon_1.$$

Proof: Consider a system:

$$\begin{cases} \frac{dS}{dt} = A_1 - (\nu + d_1)S \\ \frac{dV}{dt} = \nu S - d_1 V \\ \frac{dS_d}{dt} = A_2 - d_2 S_d \end{cases} \quad (6)$$

It is easy to see that system (6) has a unique positive equilibrium (S^0, V^0, S_d^0) which is globally asymptotically stable.

Since $R_0 > 1 \Leftrightarrow s(M) > 0$, choose small enough $\varepsilon > 0$ such that $s(M_2) > 0$, where $M_2 = M - \varepsilon M_0$,

$$M_0 = \begin{pmatrix} \beta & \phi & 0 \\ 0 & 0 & 0 \\ 0 & \phi_d & 0 \end{pmatrix}$$

Next consider a perturbed system:

$$\begin{cases} \frac{dS}{dt} = A_1 - (\nu + d_1)S - \varepsilon_1 S(\beta + \phi) \\ \frac{dV}{dt} = \nu S - d_1 V \\ \frac{dS_d}{dt} = A_2 - d_2 S_d - \varepsilon_1 S_d \phi_d \end{cases} \quad (7)$$

Because the positive equilibrium of system (6) is globally asymptotically stable, choose small enough $\varepsilon_1 > 0$ such that system (7) exist a unique positive equilibrium $(S^0(\varepsilon_1), V^0(\varepsilon_1), S_d^0(\varepsilon_1))$ which is globally asymptotically stable. $S^0(\varepsilon_1)$ and $S_d^0(\varepsilon_1)$ are continuous in ε_1 , we can restrict ε_1 small enough such that $S^0(\varepsilon_1) > S^0 - \varepsilon$ and $S_d^0(\varepsilon_1) > S_d^0 - \varepsilon$.

Suppose Theorem 4.1 is not true, then there is a $T > 0$ such that $I(t) < \varepsilon_1, W(t) < \varepsilon_1, I_d(t) < \varepsilon_1$, for all $t \geq T$. When $t \geq T$,

$$\begin{cases} \frac{dS}{dt} \geq A_1 - (\nu + d_1)S - \varepsilon_1 S(\beta + \phi) \\ \frac{dV}{dt} = \nu S - d_1 V \\ \frac{dS_d}{dt} \geq A_2 - d_2 S_d - \varepsilon_1 S_d \phi_d \end{cases} \quad (8)$$

Because the equilibrium of system (7) is globally asymptotically stable and $S^0(\varepsilon_1) > S^0 - \varepsilon, S_d^0(\varepsilon_1) > S_d^0 - \varepsilon$. Then exists a $T_1 > T > 0$ such that $S(t) > S^0 - \varepsilon, S_d(t) > S_d^0 - \varepsilon$ for $t > T_1$. Therefore, for $t > T_1$, we have

$$\begin{cases} \frac{dI}{dt} \geq (\beta I + \phi W)(S^0 - \varepsilon) - (d_1 + \alpha)I \\ \frac{dW}{dt} = kI - (\mu + n\tau)W + \lambda I_d \\ \frac{dI_d}{dt} \geq \phi_d W(S_d^0 - \varepsilon) - (d_2 + c)I_d \end{cases} \quad (9)$$

Considering the following system:

$$\begin{cases} \frac{dI'}{dt} = (\beta I' + \phi W')(S^0 - \varepsilon) - (d_1 + \alpha)I' \\ \frac{dW'}{dt} = kI' - (\mu + n\tau)W' + \lambda I'_d \\ \frac{dI'_d}{dt} = \phi_d W'(S_d^0 - \varepsilon) - (d_2 + c)I'_d \end{cases} \quad (10)$$

Because $s(M) > 0$, it's easy to see that $(I'(t), W'(t), I'_d(t)) \rightarrow (\infty, \infty, \infty)$ as $t \rightarrow \infty$. Using the comparison principle of Smith and Waltman [21], we can draw the conclusion that $(I(t), W(t), I_d(t)) \rightarrow (\infty, \infty, \infty)$ as $t \rightarrow \infty$, which leads to a contradiction. Thus, we conclude that

$$\limsup_{t \rightarrow \infty} \max\{I(t), W(t), I_d(t)\} > \varepsilon_1.$$

Theorem 4.2 If $R_0 > 1$, then system (2) admits at least one positive equilibrium and there is a positive constant ε such that every solution $(S(t), I(t), V(t), W(t), S_d(t), I_d(t))$ of the system (2) with $((S(0), I(0), V(0), W(0), S_d(0), I_d(0))) \in X_0$

$$\min\{\liminf_{t \rightarrow \infty} I(t), \liminf_{t \rightarrow \infty} W(t), \liminf_{t \rightarrow \infty} I_d(t)\} > \varepsilon,$$

which implies that the system (2) is uniformly persistent.

Proof: Consider system (2), it is easy to see that both X and X_0 are positively invariant and ∂X_0 is relatively closed in X . In addition, system (2) is point dissipative.

Define $\Omega_\partial = \{(S(0), I(0), V(0), W(0), S_d(0), I_d(0)) \mid (S(t), I(t), V(t), W(t), S_d(t), I_d(t)) \in \partial X_0, \forall t \geq 0\}$.

Now we will prove that

$$\Omega_\partial = \{(S(0), 0, V(0), 0, S_d(0), 0) \mid S(t), V(t), S_d(t) \geq 0\}.$$

It is easy to see that

$$\{(S(0), 0, V(0), 0, S_d(0), 0) \mid S(t), V(t), S_d(t) \geq 0\} \subseteq \Omega_\partial,$$

thus we just only need to prove the following:

$$\Omega_\partial \subseteq \{(S(0), 0, V(0), 0, S_d(0), 0) \mid S(t), V(t), S_d(t) \geq 0\} \quad (11)$$

As $(S(0), I(0), V(0), W(0), S_d(0), I_d(0)) \in \Omega_\partial$, we need to prove that $I(t) = 0, W(t) = 0, I_d(t) = 0$ for all $t \geq 0$. If is not true, then there exist a $t_0 \geq 0$ such that one of the following holds:

$$(i) I(t_0) > 0, (ii) W(t_0) > 0, (iii) I_d(t_0) > 0.$$

If the case (i) holds, then from the second equation of system (2), we have:

$$\frac{dI(t)}{dt} \geq -(d_1 + \alpha)I(t),$$

then

$$I(t) \geq I(t_0)e^{-(d_1+\alpha)(t-t_0)} > 0, \forall t \geq t_0$$

Further, from the fourth equation of system 2, we have

$$\frac{dW(t)}{dt} > -(\mu + n\tau)W(t), \forall t \geq t_0,$$

it follows

$$W(t) > W(t_0)e^{-(\mu+n\tau)(t-t_0)} \geq 0, \forall t \geq t_0$$

Finally, from the sixth equation of system (2), we also have

$$\frac{dI_d(t)}{dt} > -(d_2 + c)I_d(t), \forall t \geq t_0,$$

this gives

$$I_d(t) > I_d(t_0)e^{-(d_2+c)(t-t_0)} \geq 0, \forall t \geq t_0$$

Thus, for all $t > t_0$, we have $(I(t), W(t), I_d(t)) > 0$. So $(S(t), I(t), V(t), W(t), S_d(t), I_d(t))$ does not belong to ∂X_0 , for $t > t_0$, which is a contradiction. For the cases (ii) and (iii), one can also have the similar contradictions, this concludes equality (11) holds.

Since P_0 is globally asymptotically stable for system (2), and it is the only equilibrium in Ω_∂ , by afore-mentioned claim, it then follows that P_0 is isolated invariant set in X , $W^s(P_0) \cap X_0 = \emptyset$. Clearly, every orbit in Ω_∂ converges to P_0 , P_0 is acyclic in Ω_∂ . Using Theorem 4.6 in Thieme [22], we conclude that system (2) is uniformly persistent with respect to $(X_0, \partial X_0)$. By Theorem 2.4 in Zhao [23], system (2) has an equilibrium $(S^*, I^*, V^*, W^*, S_d^*, I_d^*) \in X_0$. We further claim that $S^*, V^*, S_d^* > 0$. Suppose that $S^* = V^* = S_d^* = 0$, from system (2), it can be seen that $I^* = W^* = I_d^* = 0$. This contradiction proves that $P^* = (S^*, I^*, V^*, W^*, S_d^*, I_d^*)$ is a positive equilibrium of system (2).

Theorem 4.3 The endemic equilibrium P^* of system (2) is globally asymptotically stable when $R_0 > 1$.

Proof: Define a Lyapunov function as follows:

$$\begin{aligned} L_2 = & b_1(S - S^* - S^* \ln \frac{S}{S^*}) + b_2(I - I^* - I^* \ln \frac{I}{I^*}) \\ & + b_5(S_d - S_d^* - S_d^* \ln \frac{S_d}{S_d^*}) + b_6(I_d - I_d^* - I_d^* \ln \frac{I_d}{I_d^*}) \\ & + b_3(V - V^* - V^* \ln \frac{V}{V^*}) + b_4(W - W^* - W^* \ln \frac{W}{W^*}) \end{aligned}$$

where

$$\begin{aligned} b_1 = b_2 = b_3 = & \frac{kI^*}{\phi W^* S^*}, \\ b_4 = 1, b_5 = b_6 = & \frac{\lambda I_d^*}{\phi_d W^* S_d^*} \end{aligned}$$

then the derivative of L_2 along with the solution of system (2) is

$$\begin{aligned} \frac{dL_2}{dt} = & b_1(1 - \frac{S^*}{S})[(A_1 - \beta SI - \nu S - \phi SW - d_1 S) \\ & - (A_1 - \beta S^* I^* - \nu S^* - \phi S^* W^* - d_1 S^*)] \\ & + b_4(1 - \frac{W^*}{W})(kI - \frac{kI^* + \lambda I_d^* W}{W^*} + \lambda I_d) \\ & + b_2(1 - \frac{I^*}{I})(\beta SI + \phi SW - \frac{S^*(\beta I^* + \phi W^*)}{I^*} I) \\ & + b_3(1 - \frac{V^*}{V})(\nu S - \frac{\nu S^*}{V^*} V) \\ & + b_5(1 - \frac{S_d^*}{S_d})[(A_2 - \phi_d S_d W - d_2 S_d) \\ & - (A_2 - \phi_d S_d^* W^* - d_2 S_d^*)] \\ & + b_6(1 - \frac{I_d^*}{I_d})(\phi_d S_d W - \frac{\phi_d S_d^* W^*}{I_d^*} I_d) \\ \leq & b_1 \beta S^* I^* (2 - \frac{S^*}{S} - \frac{S}{S^*}) + b_1 d_1 S^* (2 - \frac{S^*}{S} - \frac{S}{S^*}) \\ & + b_1 \phi S^* W^* (\ln \frac{I}{I^*} - \frac{I}{I^*} + \frac{W}{W^*} - \ln \frac{W}{W^*}) \\ & + b_1 \nu S^* (3 - \frac{S^*}{S} - \frac{V}{V^*} - \frac{V^* S}{S^* V}) \\ & + b_4 k I^* (\frac{I}{I^*} - \ln \frac{I}{I^*}) + b_4 \lambda I_d^* (\frac{I_d}{I_d^*} - \ln \frac{I_d}{I_d^*}) \\ & + (b_4 k I^* + b_4 \lambda I_d^*) (\ln \frac{W}{W^*} - \frac{W}{W^*}) \\ & + b_5 d_2 S_d^* (2 - \frac{S_d^*}{S_d} - \frac{S_d}{S_d^*}) \\ & + b_5 \phi_d S_d^* W^* (\frac{W}{W^*} - \ln \frac{W}{W^*} - \frac{I_d}{I_d^*} + \ln \frac{I_d}{I_d^*}) \end{aligned}$$

So, we can obtain

$$\begin{aligned} \frac{dL_2}{dt} \leq & (b_1 \beta S^* I^* + b_1 d_1 S^*) (2 - \frac{S^*}{S} - \frac{S}{S^*}) \\ & + b_5 d_2 S_d^* (2 - \frac{S_d^*}{S_d} - \frac{S_d}{S_d^*}) \\ & + b_1 \nu S^* (3 - \frac{S^*}{S} - \frac{V}{V^*} - \frac{V^* S}{S^* V}) \\ \leq & 0. \end{aligned}$$

The equation $\frac{dL_2}{dt} = 0$ holds if and only if $S = S^*, I = I^*, V = V^*, W = W^*, S_d = S_d^*, I_d = I_d^*$. Thus the endemic equilibrium P^* is globally asymptotically stable in X by LaSalle's Invariance Principle [20]. This completes the proof.

As for the subsystem composed of last two equations of system (1):

$$\begin{cases} \frac{dS_h}{dt} = A_3 - \beta_h S_h I - \phi_h S_h W - \beta_d S_h I_d + \gamma I_h - d_3 S_h \\ \frac{dI_h}{dt} = \beta_h S_h I + \phi_h S_h W + \beta_d S_h I_d - \gamma I_h - d_3 I_h \end{cases} \quad (12)$$

Using the method of limit system, we easily have the following result:

Theorem 4.4 The disease free equilibrium $(S_h^0, 0)$ of system (12) is globally asymptotically stable when $R_0 < 1$. The endemic equilibrium (S_h^*, I_h^*) of system (12) is globally asymptotically stable when $R_0 > 1$.

where $S_h^0 = \frac{A_3}{d_3}$ and

$$S_h^* = \frac{A_3(\gamma + d_3)}{d_3(\beta_h I^* + \phi_h W^* + \beta_d I_d^* + \gamma + d_3)},$$

$$I_h^* = \frac{A_3(\beta_h I^* + \phi_h W^* + \beta_d I_d^*)}{d_3(\beta_h I^* + \phi_h W^* + \beta_d I_d^* + \gamma + d_3)}$$

Therefore, we have the following result for system (1):

Theorem 4.5 The disease free equilibrium $(S^0, 0, V^0, 0, S_d^0, 0, S_h^0, 0)$ is globally asymptotically stable when $R_0 < 1$. The endemic equilibrium $(S^*, I^*, V^*, W^*, S_d^*, I_d^*, S_h^*, I_h^*)$ is globally asymptotically stable when $R_0 > 1$.

V. NUMERICAL SIMULATIONS

In this section, we carry out numerical simulations to support our theoretical results. From cases of brucellosis reported in the People’s Republic of China, we can obtain the data on human brucellosis cases in 2005 to 2014. Therefore, some parameters are come from real data [8] and some others are fitted. Taking parameter as follows: $\beta = 0.18 \times 10^{-6}, \beta_h = 1.88 \times 10^{-9}, \beta_d = 0.79 \times 10^{-8}, \phi = \phi_d = 0.1 \times 10^{-6}, \phi_h = 0.7 \times 10^{-9}, n = 0, \tau = 0, d_1 = 0.6, d_2 = 0.8, d_3 = 0.00559, k = 12, \lambda = 12, \mu = 3.6, c = 15, \gamma = 0.4$. Using system (1), we evaluate the human brucellosis data in China from 2005 to 2014 and make a prediction about the trend of human brucellosis infection. Using MATLAB, we get Figure 1, seeing that our model has reasonable parameter values and the simulation results are consistent with the national data of brucellosis infection cases from 2005 to 2014. Taking $A_1 = A_2 = 1.976 \times 10^7, \beta = 0.18 \times 10^{-6}, \phi = \phi_d = 0.1 \times 10^{-6}, \nu = 0.316, \alpha = 20, n = 0, \tau = 0, d_1 = 0.6, d_2 = 0.8, k = 12, \lambda = 12, \mu = 3.6, c = 15, \gamma = 0.4$, through numerical calculation, the basic reproduction number $R_0 < 1$. It can be seen from the Figure 2 that the density of infected populations will approach to zero, the disease-free equilibrium P_0 of system (2) is globally asymptotically stable. Changing some parameter values: $\nu = 0.316 \times 0.25, \alpha = 15$ and keep the other parameters the same as mentioned above, then the value of basic reproduction number R_0 becomes bigger than unity. It can be seen from the Figure 3 that density of infected populations will approach to a positive value. So, the endemic equilibrium point P^* of system (2) is globally asymptotically stable. At the same time, we also considered the dynamic model of the dogs direct contact with the infected sheep. The infection rate of brucella between sheep and dogs was defined as ρ . As can be seen from Figure 4, when $R_0 = 1.4587 > 1, \rho = 0.15 \times 10^{-6}$ the endemic equilibrium is globally asymptotically stable.

VI. CONCLUSION AND DISCUSSION

The spread of brucellosis has become a major concern in China and Brucella of sheep has a high infection rate to humans and other animals. Although dogs have a strong resistance to brucella infection, dogs can also cause human infection. Xinjiang is one of the five major pastoral areas in China, and there are a large number of dogs in the pastoral areas. Especially in the suburbs of Xinjiang, where are large amount of crossing areas of farming and animal husbandry, dogs also play the role of intermediate media in brucellosis transmission. The investigation on brucella Canis is of great significance for promoting the development

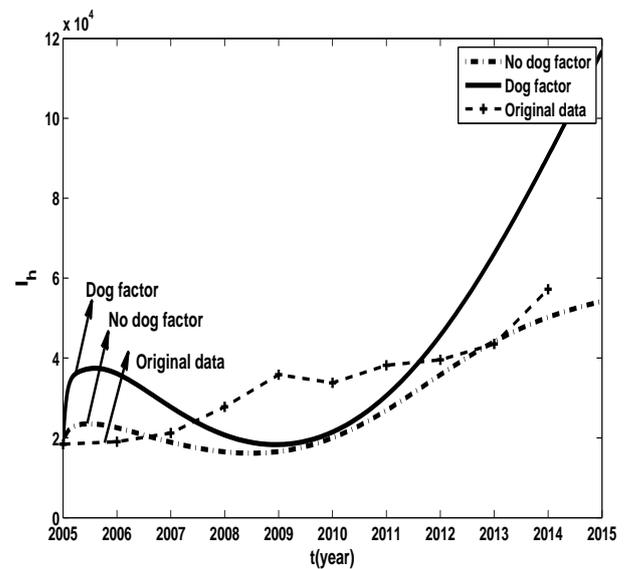


Fig. 1. A comparison of factors with and without dogs

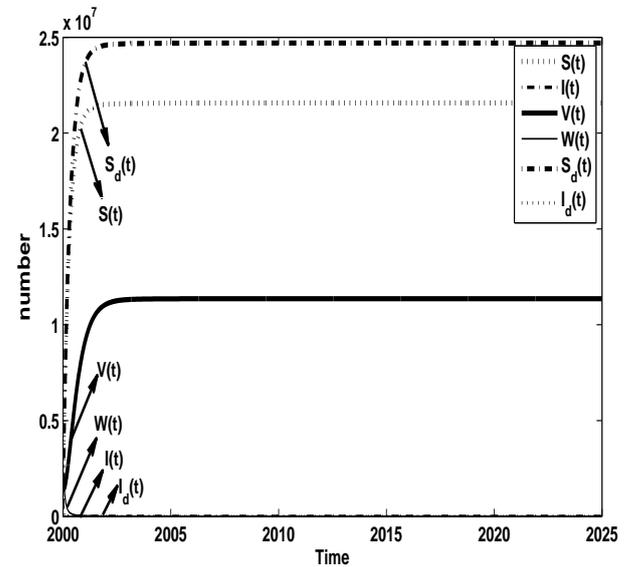


Fig. 2. When $R_0 = 0.9576 < 1$, the density of infected populations will approach to zero.

of animal husbandry and ensuring human health. Thus, we considered a brucellosis infection model with sheep-dog-human in present paper. According to the numerical simulation of model (1), it is also confirmed that dogs can play an important role in human brucellosis infection. By careful calculation, we get the basic reproduction number R_0 of the model (2). We proved the global asymptotic stability of the equilibrium points by constructing suitable Lyapunov functions. The global asymptotic behavior of model (2) is determined by the size of R_0 . If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, the endemic equilibrium is globally asymptotically stable.

Because there is no ideal therapy or drug for canine brucellosis, it is convinced that the dogs with brucellosis should be culled when detected. It is also advised that both the pet dogs and shepherd dogs should be checked regularly in daily life to avoid the harm they bring to human health.

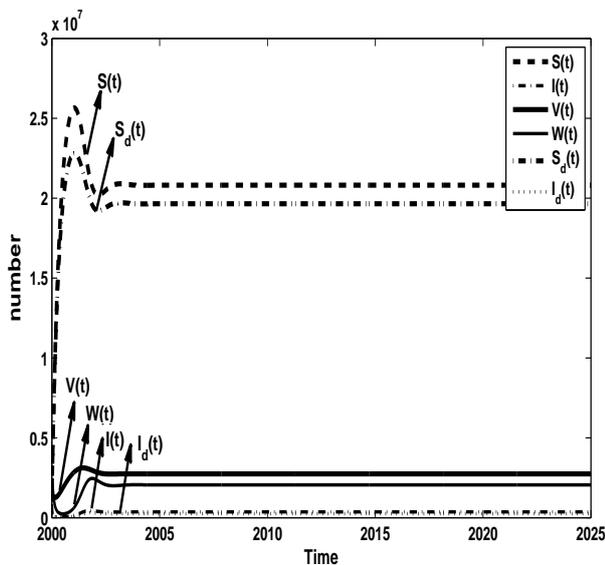


Fig. 3. When $R_0 = 1.3490 > 1$, the density of infected populations in model (2) will approach to a positive value.

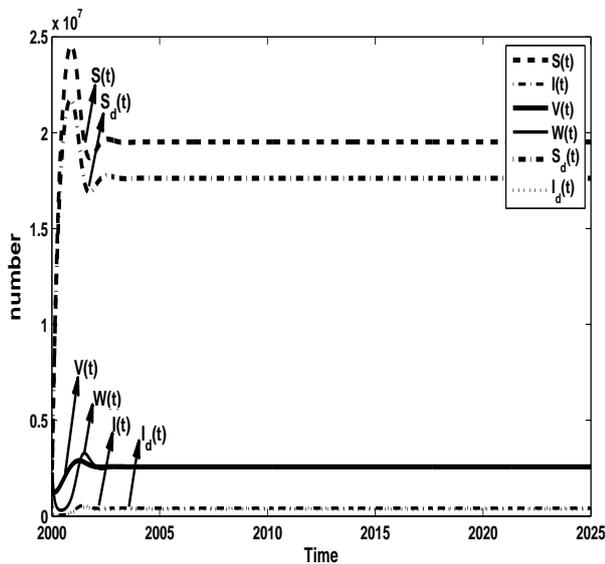


Fig. 4. When $R_0 = 1.4587 > 1$, $\rho = 0.15 \times 10^{-6}$, the endemic equilibrium of the sheep - dog direct infection model is globally asymptotically stable.

ACKNOWLEDGMENT

We are very grateful to the reviewers and handling editor for their constructive comments which improve the quality of original manuscript. We also want to express our deeply appreciation to Prof. Zhidong Teng for his many valuable suggestions.

REFERENCES

[1] Q.Hou, X.D.Sun, "Modeling sheep brucellosis transmission with a multi-stage model in Changling County of Jilin Province, China," Journal of Applied Mathematics and Computing, vol. 51, pp. 227-244, 2016.
 [2] G.Q.Sun, Z.K.Zhang, "Global stability for a sheep brucellosis models with immigration," Applied Mathematics and Computation, vol. 246, pp. 336-345, 2014.

[3] M.T.Li, G.Q.Sun and Y.F.Wu, "Transmission dynamics of a multi-group brucellosis model with mixed cross infection in public farm," Applied Mathematics and Computation, vol. 237, pp. 582-594, 2014.
 [4] K.Meng, X.Abdurahman, "Study of the brucellosis transmission with multi-stage," Communications in Mathematical Biology and Neurosciences, vol. 2018, Article ID 20, 2018.
 [5] L.H.Zhou, M.Fan, H.Xiang, Z.Jin and X.D.Sun "Transmission dynamics and optimal control of brucellosis in Inner Mongolia of China," Mathematical Biosciences and Engineering, vol. 15, n0 2, pp. 543-567, 2018.
 [6] Francesca F.Norman, Begoña Monge-Maillo, Sandra Chamorro-Tojeiro, Jose-Antonio Pérez-Molina and Rogelio López-Vélez "Imported brucellosis: A case series and literature review," Travel Medicine and Infectious Disease, vol. 14, no. 3, pp. 182-199, 2016.
 [7] D.Q.Shang, "Current status of brucella research," Chinese Journal of Control of Endemic Diseases, vol. 19, no. 4, pp. 204-212, 2004.
 [8] L.Wang, K.Wang and D.Q.Jiang, "Nontrivial periodic solution for a stochastic brucellosis model with application to Xinjiang, China," Physica A: Statistical Mechanics and its Applications, vol. 510, pp. 522-537, 2018.
 [9] T.Wang, Y.Zhang et al., "Epidemiological Investigation of Canine Brucellosis in Urumqi City of Xinjiang," China Animal Health Inspection, vol. 35, no. 6, pp. 8-11, 2018.
 [10] K.Hubbard, M.Wang, D.R.Smith, "Seroprevalence of brucellosis in Mississippi shelter dogs," Preventive Veterinary Medicine, vol. 159, pp. 82-86, 2018.
 [11] M.T.Li, G.Q.Sun and Z.Jin, "Transmission dynamics and control for a brucellosis model in Hinggan League of Inner Mongolia, China," Mathematical Biosciences and Engineering, vol. 11, no. 5, pp. 1115-1137, 2014.
 [12] M. Monir Madkour, "Madkour's Brucellosis," Springer, Berlin. 2001.
 [13] X.H.Ran, J.J.Cheng, M.M.Wang and X.H.Wang, "Brucellosis seroprevalence in dairy cattle in China during 2008-2018: A systematic review and meta-analysis," Acta Tropica, vol. 189, pp. 117-123, 2019.
 [14] P.S.Douris, M.P.Markakis, "Global Connecting Orbits of a SEIRS Epidemic Model with Nonlinear Incidence Rate and Nonpermanent Immunity," Engineering Letters, vol. 27, no. 4, pp. 866-875, 2019.
 [15] J.Nie, G.Q.Sun and J.Zhang, "Modeling the transmission dynamics of dairy cattle in Jilin province, China," Journal of Biological Systems, vol. 22, no. 4, pp. 533-554, 2014.
 [16] J.Zinsstag, F.Roth and D.Orkhon, "A model of animal-human beucellosis transmission in Mongolia," Preventive Veterinary Medicine, vol. 69, no. 1-1, pp. 77-95, 2005.
 [17] Afeez Abidemi, Mohd Ismail Abd Aziz and Rohanin Ahmad, "The Impact of Vaccination, Individual Protection, Treatment and Vector Controls on Dengue," Engineering Letters, vol. 27, no. 3, pp. 613-622, 2019.
 [18] H.Miao, X.Abdurahman, Z.D.Teng and C.J.Kang, "Global Dynamics of a Fractional Order HIV Model with Both Virus-to-Cell and Cell-to-Cell Transmissions and Therapy Effect," IAENG International Journal of Applied Mathematics, vol. 47, no. 1, pp. 75-81, 2017 .
 [19] P.Van den Driessche, J.Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," Mathematical Biosciences, vol. 180, no. 1-1, pp. 29-46, 2002.
 [20] J.P.LaSalle, "The stability of dynamical systems," Regional Conference Series in Applied Mathematics. SIAM. Philadelphia. 1976.
 [21] H.L.Smith, P.Waltman, "The Theory of the Chemostat," Cambridge University Press. 1995.
 [22] H.R.Thieme, "Persistence under relaxed point-dissipativity (with application to an endemic model)," SIAM Journal on Mathematical Analysis, vol. 24, no. 2, pp. 407-435, 1993.
 [23] X.Q.Zhao, "Uniform persistence and periodic coexistence states in infinite-dimensional periodic semiflows with applications," Canadian Applied Mathematics Quarterly, vol. 3, no. 4, pp. 473-495, 1995.